"ANALYSIS OF PRESENTATION, MANAGEMENT AND OUTCOME OF CHRONIC CALCIFIC PANCREATITIS IN A TERTIARY CARE CENTER "

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CERTIFICATE

This is to certify that this dissertation entitled **"ANALYSIS OF PRESENTATION, MANAGEMENT AND OUTCOME OF CHRONIC CALCIFIC PANCREATITIS IN A TERTIARY CARE CENTER "** presented here is original work done by **Dr.R.RAJKUMAR,** M.Ch Post Graduate in the Department of Surgical Gastroenterology and Proctology, Center of Excellence for Upper GI Surgery, Madras Medical College and Rajiv Gandhi Government General Hospital, Chennai- 600003 in partial fulfillment of the university rules and regulation for the award of M.Ch in Surgical Gastroenterology and Proctology-Branch VI, under my guidance and supervision during the academic period from August 2011- March 2014.

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DECLARATION

I, **Dr.R.RAJKUMAR** hereby solemnly declare that this dissertation entitled **"ANALYSIS OF PRESENTATION, MANAGEMENT AND OUTCOME OF CHRONIC CALCIFIC PANCREATITIS IN A TERTIARY CARE CENTER "** was done by me in the Department of Surgical Gastroenterology and Proctology, Center of Excellence for Upper GI Surgery, Madras Medical College & Rajiv Gandhi Govt. General Hospital, Chennai-3 during August 2011 to March 2014 under the guidance and supervision of **Prof. S.M.CHANDRAMOHAN M.S,M.ch, FACS.** This dissertation is submitted to the Tamil Nadu Dr.M.G.R.Medical University towards the partial fulfillment of requirement for the award of M.Ch.,Degree in Surgical Gastroenterology.

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INDEX

S.NO	CONTENTS	PAGE NO.
1.	INTRODUCTION	1
2.	AIMS AND OBJECTIVES	3
3.	REVIEW OF LITERATURE	4
4.	MATERIALS AND METHODS	27
5.	OBSERVATION AND RESULTS	29
6.	DISCUSSION	45
7.	CONCLUSION	48
8.	BIBLIOGRAPHY	50
9.	ANNEXURE A) PROFORMA B) PATIENT INFORMATION SHEET C) MASTER CHART D) ETHICAL COMMITTEE APPROVAL ORDER E) PLAGIARISM	59

Abstract

"Analysis Of Presentation, Management And Outcome Of Chronic Calcific Pancreatitis In A Tertiary Care Center "

Introduction

Chronic calcific pancreatitis is a commonly encountered disease in South India. The presentation of these patients include pain, steatorrhea, diabetes mellitus, UGI bleed due to Gastric varices, Ascites, jaundice, pseudocyst and rarely as duodenal obstruction

Aim and background:

To analyse the demographics, spectrum of clinical presentation ,indications for surgery, type of surgery, intra operative variables peri operative complications and outcomes of surgery**.**

Methods

This is a retrospective study of 80 cases of Chronic calcific pancreatitis who had surgery in our department. The study included the following parameters: Age of presentation, Gender, Etiological factors, Chief symptoms, duration of symptoms, various diagnostic modalities used for diagnosis of the disease and the different types of complications on presentation. We also included the details of preoperative work up we made and the preoperative treatments given. Also we made the analysis of the different types of drainage procedures, resection procedures and other additional procedures done for the complicated cases. We analysed the intra op and post operative complications and also analysed the short term follow up results of the 50 patients after discharge. This study was done between August 2011- March2014

Results

Based on this study we found that this disease is commonly seen in males(75%), Ethanol induced pancreatitis was found to be the predominant cause(72.5%) and common age group is between 30-45 years. The commonest presentation in all patients is Pain, 12.5% had diabetes mellitus and 7.5% of them had steatorrhea. We encountered following complications on evaluation- three patient had pancreatic ascites, three patients had splenic vein thrombosis and portal hypertension, seven patients had Pancreatic pseudocyst and ten patients had jaundice.

Freys head coring pancreatico jejunostomy was commonly done by us in about 68 patients, one of whom had pancreatic pseudocyst underwent cysto gastrostomy, 2 patients with small duct disease underwent Izbecki procedure of making V shaped incision in duct and ten patients underwent Lateral pancreatico jejunostomy. Five of them underwent distal pancreatectomy & Splenectomy as additional procedure (3 for splenic vein thrombosis, 1 for pancreatico pleural fistula 1 for infected pancreatic necrosis at tail). One patient Choledocho duodenostomy as additional procedure. It has been studied that Frey's Head Coring Pancreatico jejunostomy provided better quality of pain relief and quality of life.

Conclusion

This study enlightens that, freys procedure done by single layer anastomosis is cost effective, less time consuming and showing no difference in terms of post operative complications like pancreatic leak with zero mortality.

INTRODUCTION

Chronic pancreatitis (CP) is a progressive inflammatory disorder characterized by irreversible destruction of pancreatic parenchyma,associated with disabling chronic pain and permanent loss of exocrine and endocrine function. Although the disease was first described by Friedr1ich in 1878, there is yet incomplete understanding of the pathophysiology and natural history of the disease.

The management of patients with chronic pancreatitis remains a challenge because of the limited understanding of the pathophysiological process of the disease, the unpredictability of clinical evo1lution and the controversies between diagnostic criteria and therapeutic options. Worldwide the main aetiological factor is alcohol abuse, and the most common symptom is relentless chronic abdominal pain. [1]

After optimization of symptoms with analgesics and enzyme supplementation, patients with persistent symptoms are candidates for invasive treatments. Previous studies have shown that surgical treatment of chronic pancreatitis reduces pain and subsequent complications, so that patients return to their prior work activities as well as improved quality of life. The diseases

assumes greater significance on account of the fact it affects young men and women in the prime of their lives.

Historically, the surgical procedures have been classified into two categories:

(i) drainage

(ii) resection.

In recent decades, procedures combining resection and drainage have evolved, such as the FREYS procedure.

The aim of this retrospective study was to describe a 3-year experience using Frey's procedure in a consecutive series of patients from a single institution.

AIMS AND OBJECTIVES

To analyse the demographics, spectrum of clinical presentation ,indications for surgery, type of surgery, intra operative variables peri operative complications and outcomes of surgery.

REVIEW OF LITERATURE

INTRODUCTION

Chronic pancreatitis is a progressive inflammatory disorder that leads to irreversible destruction of the exocrine and endocrine tissue of the pancreas. Fibrotic replacement of the normal pancreas may be associated with persistent abdominal pain, the development of exocrine insufficiency, and ultimately, diabetes mellitus. Inflammation may lead to local complications, including biliary and gastrointestinal obstruction, ascites, mesoportal-splenic thrombosis, pseudocyst formation, hemorrhage, and sepsis. Despite multiple consensus conferences, there are no uniformly satisfactory criteria for the diagnosis, classification, and staging of chronic pancreatitis. In its advanced stages, chronic pancreatitis is readily apparent clinically, typically associated with pancreatic duct stricture and ductal dilation, stones and diffuse parenchymal calcification, and the digestive and metabolic effects of organ insufficiency. However, recognition of patients with early and mild disease remains a difficult challenge. The absence of a clinically relevant classification system for chronic pancreatitis contributes to inconsistencies in the treatment of the disease. Treatment decisions, ideally taken after appropriate multidis-ciplinary input from surgical, endoscopic, and radiologic experts, are better made in the context of individual circumstances such as patient symptoms and

anatomic findings rather than classification systems based on etiology or morphologic severity.

DEFINITION: CHRONIC PANCREATITIS

. Chronic pancreatitis generally refers to an ongoing inflammatory and fibrosing disorder characterized by irreversible morphologic changes, progressive and permanent loss of exocrine and endocrine function, and a clinical pattern of either recurrent acute exacerbation or persistent pain. In reality, however, acute and chronic pancreatitis represent more of a spectrum of inflammatory and fibrosing conditions of the pancreas than the two dichotomous terms would otherwise imply. Recurrent episodes (or even a single episode) of acute pancreatitis may lead to chronic changes within the pancreas, although the timing and extent to which such changes merit a change in nomenclature to chronic pancreatitis is somewhat arbitrary. The histopathologic changes of chronic pancreatitis comprise fibrosis, a reduced number of acinar cells and islets of Langerhans, and development of strictures and dilation of pancreatic ducts as well as calcium calculi (pancreatic duct stones). The morphologic/structural changes of chronic pancreatitis can occur years before any clinical symptoms are present. One hypothesis envisions the activation of pancreatic stellate cells, which induce desmoplasia, as the key pathogenetic "switch" that leads to the transition to chronic pancreatitis[1].

Efforts to establish consensus for uniform terminology of pancreatitis began with an international conference held in Marseille in 1963, during which participants agreed that acute pancreatitis should refer to acute organ inflammation characterized by interstitial edema, peri-pancreatic fat necrosis, and hemorrhage, which resolves if the primary cause or risk factor is removed. Chronic pancreatitis was distinguished by irreversible focal, seg-mental, or diffuse destruction of the exocrine tissue along with dilation or focal strictures of the main pancreatic duct. However, these definitions failed to address the functional or clinical dimensions of acute or chronic pancreatitis. At a second meeting in Marseille held in 1984 [23], the Chronic pancreatitis was subclassified as chronic pancreatitis with focal or segmental or diffuse fibrosis, and chronic pancreatitis with or without stones, and obstructive chronic pancreatitis was listed as a distinct form. Patients with chronic pancreatitis present with recurrent episodes of pain or with painless progressive loss of exocrine or endocrine insufficiency. To help define changes associated with clinical risk factors, a 1988 meeting in Rome added the morphologic distinction of chronic calcifying pancreatitis that is characterized by intraductal calcifications and protein plugs, and chronic inflammatory pancreatitis that is characterized by dense infiltration of mononuclear inflammatory cells [4]. A consensus conference in Cambridge in 1984 defined the distinction between acute and chronic pancreatitis as the reversibility of the morphologic and functional changes of inflammation[5.] The Cambridge meeting proposed a classification system of chronic pancreatitis

based on radiographic findings on endoscopic retrograde cholangiopancreatography (ERCP) and ultrasonography (US) or computed tomography (CT). Criteria for chronic pancreatitis from the Japan Pancreas Society focused on findings from an array of diagnostic approaches including US, CT, ERCP, secretin stimulation, and histologic examination of pancreatic tissue. The presence of certain criteria such as pancreatic stones on CT or US is considered definitive evidence of chronic pancreatitis, whereas others such as pancreatic deformity with irregular contour are considered only probable or possible evidence in support of the disease [10].

RISK FACTORS

The pathogenesis of chronic pancreatitis remains poorly understood. Most hypotheses are more associative and speculative than mechanistic. An association between alcohol and acute and chronic pancreatitis has been noted for over half a century. Sarles et al demonstrated that the relative risk of chronic pancreatitis increases directly with mean daily alcohol consumption[4.] However, even relatively moderate alcohol intake can cause chronic pancreatitis, and the duration of alcohol consumption can be relatively short before the onset of disease. In alcohol-induced acute pancreatitis, it has been postulated that by-products of alcohol metabolism induce acinar cell injury, which produces pancreatic enzyme (trypsin, chymotrypsin, and phospholipase A) activation and local tissue damage by autodigestion and recruitment of inflammatory cells. In

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chronic pancreatitis, alcohol is thought to increase the protein concentration in pancreatic juice, which causes intraductal calcium stone formation, ductal epithelial ulceration, inflammation, and fibrosis. Yet it is only a small percentage (5% to 10%) of alcoholics that develop pancreatic disease, suggesting that alcohol is more of a risk factor than a causative agent for pancreatitis in patients who are susceptible for various unknown or poorly defined reasons. Different disease processes causing similar-appearing injury to the pancreas may follow different clinical courses. Thus, rather than classifying pancreatitis based on the presumed causative agent, Whitcomb et al proposed a system to classify risk factors that may interact to predispose an individual patient to produce pancreatitis.[7] According to this framework, risk factors are grouped as Toxic/Metabolic, Idiopathic, Genetic, Autoimmune, Recurrent Acute, and Obstructive

TOXIC/METABOLIC

Almost 70% of chronic pancreatitis cases are associated with chronic alcoholic intake in Western countries.[8] The prevalence is significantly higher in men compared with women. Tobacco use is associated with the early presentation of alcoholic chronic pancreatitis and is associated with the presentation of calcifications and the development of diabetes. It is unknown whether tobacco

initiates the disease[9]; however, tobacco is thought to potentiate the progression of chronic pancreatitis. In a preclinical model, investigators demonstrated that tobacco exposure increases the risk of pancreatic cancer in chronic pancreatitis patients.[10] Hyperparathyroidism and hyper-calcemia are also associated with chronic pancreatitis. Pancreatitis has been reported to be the first manifestation of patients with multiple endocrine neoplasia 2A (MEN2A). Patients with chronic renal failure have a higher risk of chronic pancreatitis. Certain medications, including statins, steroids, oral contraceptives, and inter-feron, as well as clinical hyperlipidemia can be associated with chronic pancreatitis, but they are more often associated with acute or recurrent acute pancreatitis in high-risk patients, including the elderly and patients with cancer.

IDIOPATHIC

Historically, no environmental or metabolic risk factor can be identified in approximately 20% of patients who are therefore categorized as having idiopathic acute, recurrent acute, or chronic pancreatitis. Patients with idiopathic disease typically fall into a bimodal age distribution, presenting either between the ages of 10 and 20 or after age 50 years. However, many of these patients are increasingly recognized to have underlying genetic mutations and polymorphisms and may be more appropriately recategorized into the genetic subgroup.

GENE MUTATIONS

Under physiologic conditions, pancreatic enzyme activation is strictly controlled. Mutations in proteins that regulate this activation increase the risk of chronic pancreatitis. Mutations in the cationic trypsinogen gene (also known as protease serine 1 [PRSS1] gene) are common in hereditary chronic pancreatitis. PRSS1 is located in chromosome 7 and regulates trypsinogen production; mutations in this gene are associated with intraacinar trypsinogen activation.PRSS1mutations have been documented inhereditary pancreatitis but are uncommon in other forms of chronic pancreatitis.

SPINK-1 is a peptide secreted by acinar cells that regulates the premature activation of trypsinogen. Because SPINK1 mutations are present in 1% to 2% of healthy patients ,but the prevalence of chronic pancreatitis is much lower, it has been hypothesized that SPINK1 mutations are not enough to trigger pancreatic inflammation. However, they lower the threshold to develop it and influence the severity of the disease. SPINK1 mutations are more prevalent in alcoholic, hereditary, and idiopathic pancreatitis.

The secretion of bicarbonate and chloride in respiratory and pancreatic secretions is regulated by the CFTR gene. CFTR mutations affect the normal secretion of bicarbonate, decrease pancreatic juice volume, and augment the concentration of pancreatic enzymes inside the pancreatic duct. Homozygous

CTFR mutation result in cystic fibrosis; heterozygous mild mutations predispose to pancreatic exocrine insufficienc and chronic pancreatitis.

The prevalence of CFTR gene mutations is higher in patients with alcoholic, idiopathic, and hereditary pancreatitis as compared with the general population

AUTOIMMUNE

Autoimmune pancreatitis, also known as lymphoplasma- cytic sclerosing pancreatitis, is a rare cause (1%) of chronic pancreatitis.[21] Gland enlargement, diffuse duct narrowing, and stenosis of the intrapancreatic portion of the bile duct characterize the disease. Histologic examination of the tissue demonstrates pancreas parenchyma infiltrated by both CD4+ and CD8+ lymphocytes and IgG4 plasma cells, with interstitial fibrosis and acinar cell atrophy. Patients with autoimmune pancreatitis have antibodies directed against a peptide that is homologous with the sequence of the plasminogen-binding protein (PBP) of *Helicobacter pylori* and with the ubiquitin-protein ligase E3 component n-recognin 2 which is expressed in the acinar cells of the pancreas.[22] Autoimmune pancreatitis can be associated with other autoimmune diseases including Sjogren syndrome, primary sclerosing cholangitis (PSC), and inflammatory bowel disease. Primary treatment for autoimmune pancreatitis is steroid treat-ment. Focal inflammation seen with this disease can often mimic a pancreatic mass, which may be difficult to differentiate from a pancreatic malignancy on imaging studies.

RECURRENT ACUTE

Recurrent episodes of acute pancreatitis of any etiology can cause chronic pancreatitis. This mechanism is poorly understood but likely involves the accumulated effects of postinflammatory scarring and necrosis as well as the priming of pancreatic stellate cells to induce fibrosis. In addition, radiation and ischemia may contribute to irreversible histopathologic changes and inflammation characteristic of chronic pancreatitis.

OBSTRUCTION

Obstructive pancreatitis can be congenital, functional, or acquired. Causes of pancreatic obstruction include pancreatic or ampullary tumors, and postinjury pancreatic duct fibrosis. Elevated basal pressures at the sphincter of Oddi are thought by some to lead to relative outflow obstruction from the proximal duct and thereby contribute to pancreatitis, although direct evidence for this hypothesis is lacking and no convincing mechanism for progression to chronic disease has been proposed. Patients may also have anatomic variations in the pancreatic ductal system that predispose for obstruction, most notably pancreas divisum. Pancreas divisum occurs when the ventral and dorsal aspects of the pancreatic ducts fail to fuse during development and, consequently, the drainage of the pancreatic body and tail occurs through the dorsal duct and the minor papilla. Insufficient caliber of the dorsal duct is thought to induce relative

obstruction to outflow that could lead to pancreatitis. However, the vast majority of patients with pancreas divisum are asymptomatic; thus, the anatomic variation may predispose to pancreatitis in combination with other risk factors rather than initiating pancreatitis directly.

CLINICAL MANIFESTATIONS

The most common symptom of chronic pancreatitis is abdominal pain (90%), although the pattern of pain is highly variable. In some patients, particularly early in the course of the disease, pain may be a minor feature. The pain may be episodic and minimal or absent in between acute exacerbations, but it often is noted to gradually become more constant. In late phases of the disease, pain may disappear ("burnout"), a transition that is often associated with the development of diabetes and exocrine insufficiency. The pain is most frequently localized to the epigastrium, often radiates to the back, and is typically associated with nausea and vomiting. Overall, the course of chronic pancreatitis is highly unpredictable and variable. Because eating can exacerbate pain, patients may avoid regular meals, leading to weight loss and malnutrition. Between 4% and 30% of patients have significant exocrine insufficiency and report bloating, flatulence, or steatorrhea (foul smelling, oily, and loose stools). Malabsorption leads to weight loss and deficiencies in micronutrients, especially fat-soluble vitamins A, D, and E. Endocrine insufficiency or diabetes mellitus develops later in the course of the disease, typically when 90% of the

parenchyma is replaced by fibrosis. Diabetes develops more often in those patients with alcohol-associated chronic calcifying pancreatitis than in hereditary forms of the disease.

EXTRAPANCREATIC COMPLICATIONS OF CHRONIC PANCREATITIS

A subset of patients develops symptoms of gastrointestinal and biliary obstruction. Duodenal, colonic, and bile duct obstruction can occur as a result of significant fibro-sis of the head of the pancreas or the development of large pseudocysts (Figure 89-1). The incidence of biliary obstruction is approximately 3% to 23% among patient diagnosed with chronic pancreatitis, and is even higher (15% to 60%) among patients who require surgery.24 The incidence of duodenal obstruction/stenosis is 2% in all patients, and again is higher (12%) in patients who require operative therapy. The majority of patients with splenic vein thrombosis are asymptomatic; the incidence of thrombosis varies anywhere from 4% to 45% depending on the population surveyed, but very few patients present with gastric variceal bleeding.[25,26]

Epidemiologic and preclinical studies demonstrate that chronic pancreatitis is associated with the development of pancreatic cancer. Lowenfels et al presented a multicenter historical cohort study of 2015 patients with chronic pancreatitis followed for at least 2 years.[27] The standardized incidence risk ratio for the

development of pancreatic cancer was 16.5 and 14.4 at 2 and 5 years' followup, respectively, for the risk of developing pancreatic cancer. The incidence of pancreatic cancer was equally high in patients who presented with pancreatitis associated with chronic alcohol use and those with other risk factors.

MECHANISM OF PAIN IN CHRONIC PANCREATITIS

A number of mechanisms have been proposed to account for the pain of chronic pancreatitis. Obstruction of the main pancreatic duct in some circumstances is thought to lead to increased ductal pressure (that may in turn be transmitted to secondary ducts and the surrounding parenchyma), leading to pain through stretch-activated neural pathways. Ductal obstruction may also induce missorting and mistargeted basolateral secretion of pancreatic enzymes, triggering protease-activated nociceptive pathways. Relief of main duct obstruction via decompressive surgical procedures is often effective treatment for pain in these circumstances but is far from universally successful either in the short run or in extended followup. Chronic inflammation of the pancreas may lead to fibrosis of the peripancreatic capsule and perilobular parenchyma, which has been proposed to impair regional and local blood flow, thereby producing pain through ischemia and consequent tissue acidosis.[29] Parenchymal fibrosis has also been likened to a "compartment syndrome" of sorts, associated with impaired venous drainage.30 Chronic inflammation associated with

chronic pancreatitis may also induce visceral hyperalgesia through neural remodeling of local, spinal, or central nociceptive pathways.[31,32] Superimposed on this background of uncertainty regarding the cellular, organ, and systemic basis of pain is the confounding influence of narcotic addiction that afflicts many affected individuals.

DIAGNOSIS & IMAGING STUDIES

The diagnosis of chronic pancreatitis may be challenging early in the course of the disease because the correlation between symptoms and the structural changes seen on imaging studies is poor. The most common CT findings in chronic pancreatitis include dilated pancreatic duct (68%), parenchymal atrophy (54%), and pancreatic calcifications (50%;). Other findings include peripancreatic fluid, focal pancreatic enlargement, biliary duct dilation, and irregular pancreatic parenchyma contour.

CT has a sensitivity of 56% to 95% and a specificity of 85% to 100% for the diagnosis of chronic pancreatitis. In addition to establishing the diagnosis, CT is particularly useful to assess complications, such as pancreatic duct disruption, pseudocysts, portal and splenic vein thrombosis, splenic, and pancreaticoduodenal artery pseudoaneurysms.

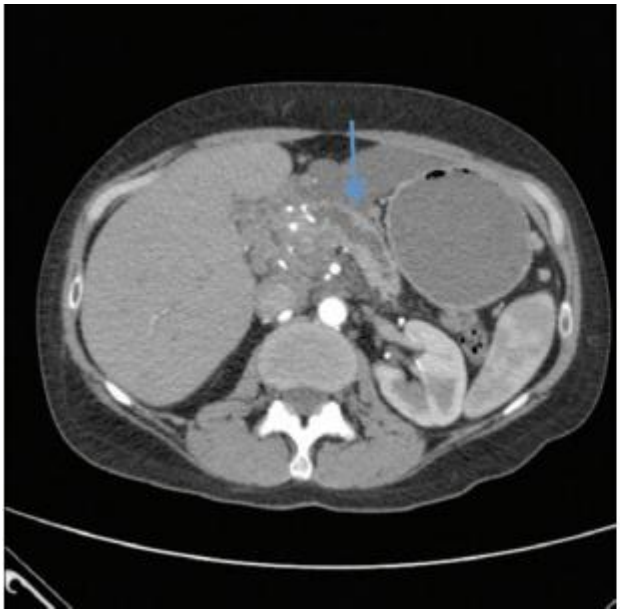


Fig1

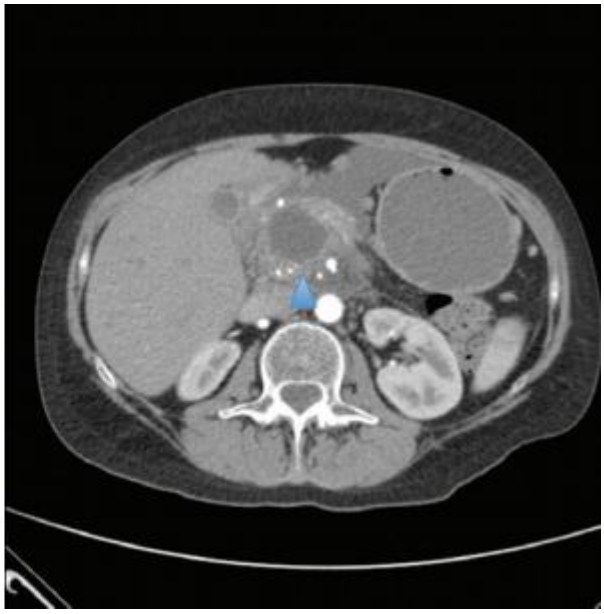


Fig 2

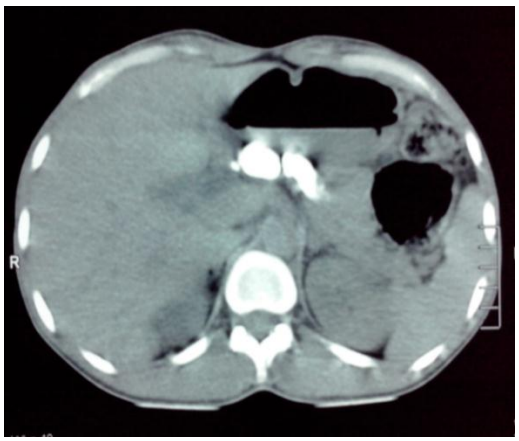


Fig3

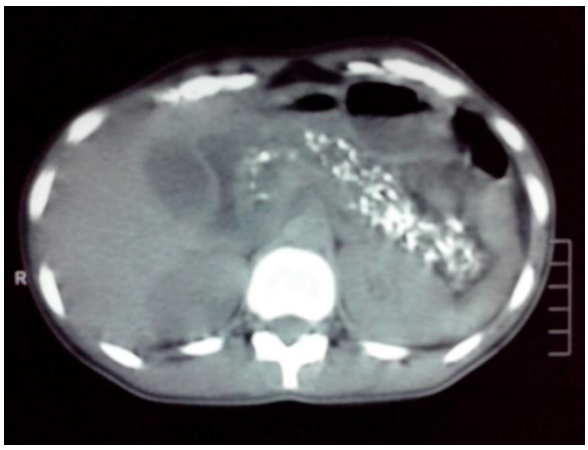
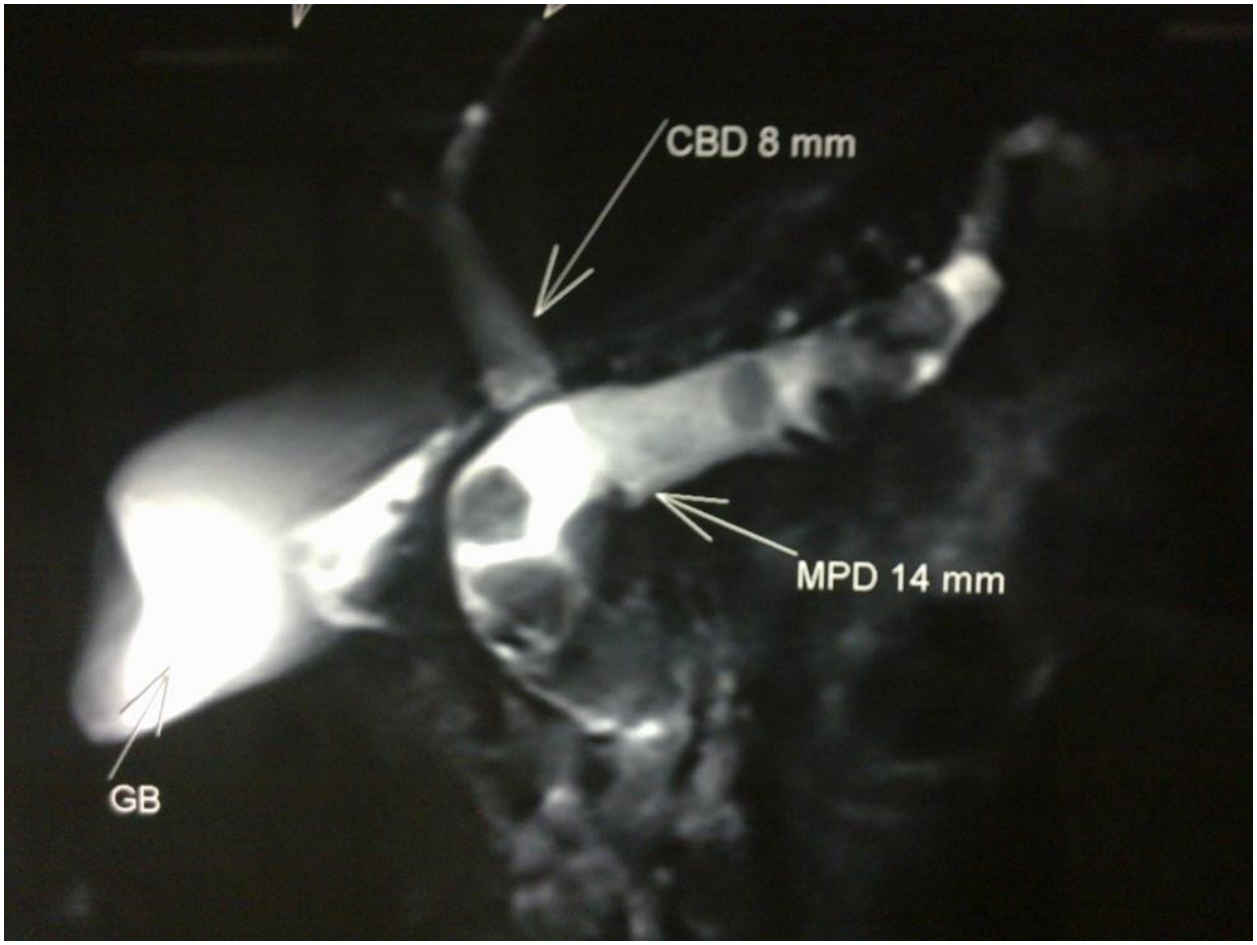


Fig4

Fig 1,2,3,4: contrast and plain CT abdomen showing calcific pancreatitis



MRI is a reliable alternative to evaluate patients with chronic pancreatitis. The sensitivity for the diagnosis of pancreatic calcifications is lower, but MRI is useful to detect changes in the pancreatic parenchyma suggestive of chronic inflammation, such as changes in intensity, pancreatic atrophy, and irregularities in the contour. In addition, MRCP with secretin injection is particularly useful to evaluate intraductal strictures and pancreatic duct disruption.

Although ERCP was historically considered the gold standard for the diagnosis of chronic pancreatitis, the advent of secretin MRCP and EUS have significantly decreased its role as a diagnostic test. Current indications include patients for whom other diagnostic tests, including CT and MRCP, are

contraindicate or have failed to corroborate the diagnosis. ERCP should be considered a therapeutic modalities in patients who develop pancreatic duct complications amenable to endoscopic therapy, such as stricture, stone ,pseudocysts, and biliary stenosis

EUS has emerged during the past 25 years as the most accurate technique to diagnose chronic pancreatitis in patients with minimal-change disease or in the early stages. Recently, a panel of endosonographers has defined the criteria required to diagnose chronic pancreatitis, known as the Rosemont criteria . Histologic evidence of inflammation, atrophy, and fibrosis is the gold standard for the diagnosis of chronic pancreatitis; however, current evidence does not support the use of EUS-guided FNA or Tru-Cut biopsies to diagnose this disease

INTERVENTIONAL THERAPY: ENDOSCOPIC TREATMENT

ERCP is the primary modality for treating symptomatic pancreatic duct obstruction with dilation and polyethylene stent placement. A number of sessions are usually required because of symptom recurrence. Note that the differential diagnosis of pancreatic duct strictures includes pancreatic cancer. Only after a thorough evaluation, which includes CT, MRCP, and/or EUS, has completely ruled out the possibility of malignancy should endoscopic treatment

be considered. Surgical resection is indicated if any concern of malignancy exists.

Endoscopic stone extraction should be considered for patients with pain and pancreatic duct dilation secondary to stones. Extracorporeal shock wave lithotripsy followed by therapeutic ERCP may be required for the treatment of large impacted stones. The success rate varies from 44% to 77% for this technique.

Biliary obstruction caused by chronic pancreatitis occurs in 10% of patients and is best treated with surgical bypass. Temporary relief of the obstruction using plastic stents is indicated for patients with cholangitis or for those who are severely malnourished. Symptomatic pseudocysts can be drained transgastrically or transdudodenally in selected patients.

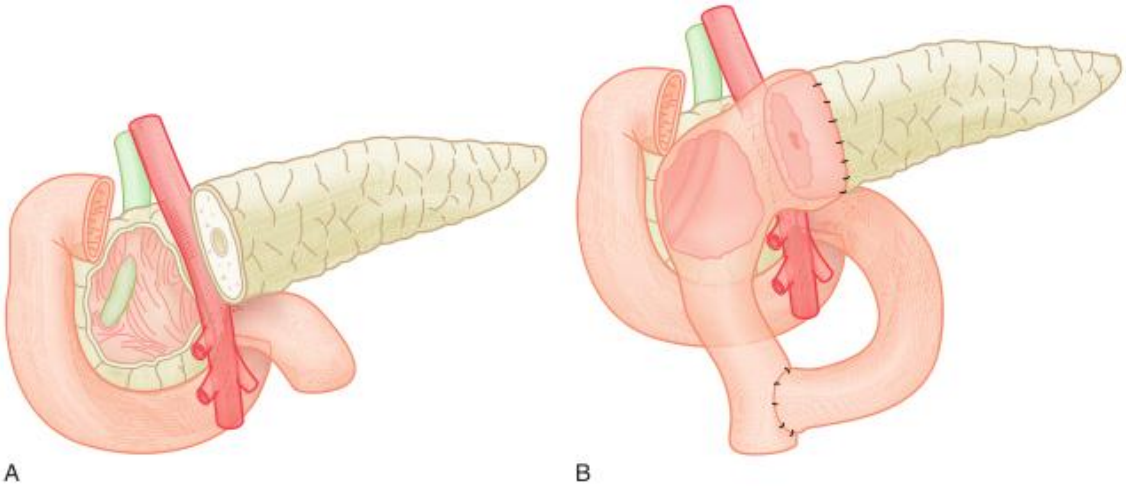
SURGICAL TREATMENT

Several factors, including intractable pain, biliary, pancreatic duct or duodenal obstruction, pseudo cyst or pseudo aneurysm formation, and the inability to rule out malignancy may prompt surgical intervention. The choice of surgical procedure depends on the symptoms requiring palliation and the presence or absence of pancreatic ductal dilation. In general, patients with a dilated pancreatic duct (defined as diameter >7 mm) require a decompressing procedure and patients with normal pancreatic duct require a resectional procedure.

RESECTION

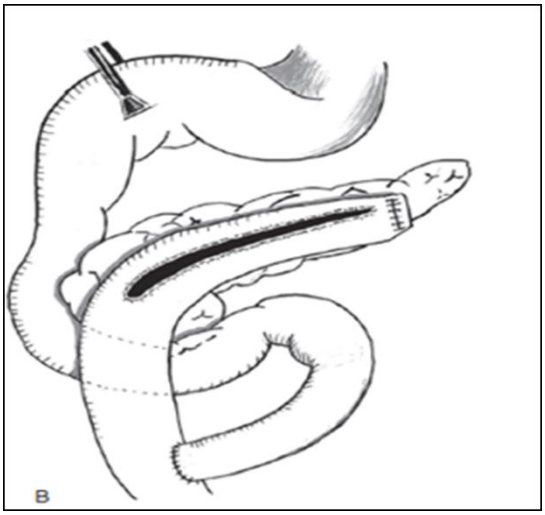
For patients with focal disease largely confined to the head of the pancreas without duct dilation, a Whipple procedure (pancreaticoduodenectomy [PD]) was generally the preferred option in olden days .[40] The procedure involves the resection of the head of the pancreas with the distal common bile duct, distal stomach, duodenum, and proximal jejunum. Removal of the head of the pancreas also addresses bile duct stricture and duodenal obstruction, if present, and improves drainage of the upstream proximal (main) pancreatic duct and its tributaries. The reconstruction after resection of the Whipple specimen includes a two-layered end-to-side pancreatico-jejunostomy , an end-to-side hepaticojejunostomy, and a gastrojejunostomy. Pain relief, either complete or partial, is usually achieved in approximately 85% of patients. Mortality associated with the procedure is generally less than 5% and near zero in experienced centers, although the overall rate of postoperative complications is relatively high, typically reported between 30% and 40%. Traverso and Longmire introduced a pylorus-preserving pancre-aticoduodenectomy (PPPD), an operation that was intended to improve functional digestive outcomes and quality of life by preserving the physiologic gastric emptying mechanism.[42] Long-term followup studies show no meaningful differences in functional outcome or maintenance of weight, and PPPD may in fact be associated with a slightly higher incidence of early postoperative delayed gastric emptying than classic PD.

Beger introduced duodenum-preserving pancreatic head resection (DPPHR) as an alternative to PD or PPPD.[43] The procedure includes division of the neck of pancreas overlying the confluence of the splenic and superior mesenteric veins and removal of the head of the pancreas, leaving a small rim of pancreatic tissue along the duodenum. The procedure is completed with end-to-end and side-to-side Roux-en-Y pancreaticojejunos-tomy (Figure A). DPPHR maintains gastrointestinal and biliary continuity, and achieves similar pain relief[43-45] and improvement in quality of life[46] as PD. Key steps in the procedure include identification and preservation of the posterior branch of the gastroduodenal artery and the intrapancreatic portion of the common bile duct. Gloor et al described a modification of the DPPHR, known as the Berne procedure, that involves excavation of the central portion of the head without formal division of the neck.[47]



DECOMPRESSION

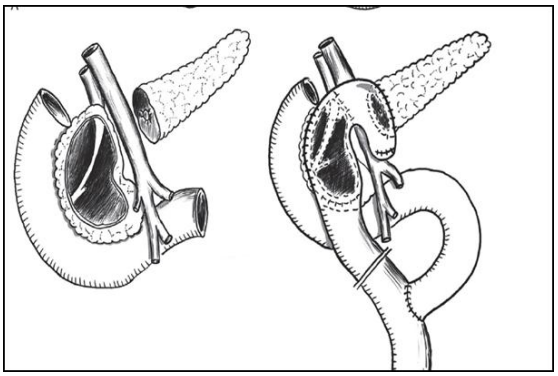
For patients with large duct disease and no focal inflammatory mass, duct-enteric drainage is the preferred treatment. In 1954, Duval described drainage of the tail of the pancreas with a Roux-en-Y limb of jejunum as a procedure for chronic pancreatitis. This operation often failed because it did not address disease in the proximal pancreas. Puestow and Gillesby introduced a modified procedure to drain the entire pancreatic duct along the body and tail of the pancreas laterally into a Roux-en-Y limb ofjejunum, which was initially described in conjunction with splenectomy and the distal pancreatectomy.[48] Partington and Rochelle simplified the Puestow technique by eliminating splenectomy and pancreatic resection.[49] The Puestow procedure or the lateral pan-creaticojejunostomy involves a retrocolic side-to-side Roux-en-Y pancreaticojejunostomy



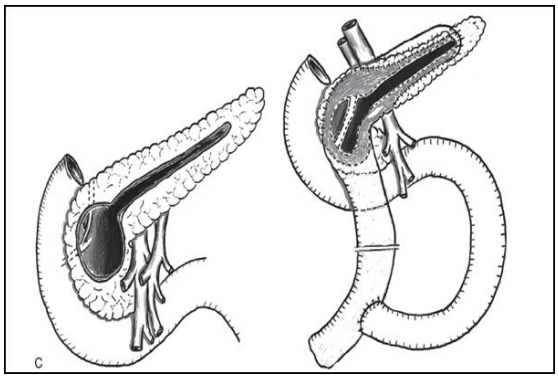
Partingtons rochalle procedure.

HYBRID PROCEDURES

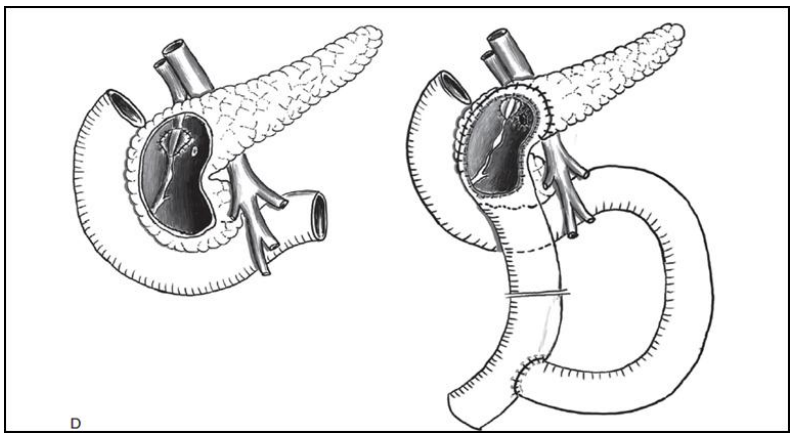
Some patients present with not only large duct disease but also significant inflammatory disease within the head of the pancreas, and Puestow-type lateral pancreaticoje-junostomy may be insufficient to address potential sources of pain within the pancreatic head.



Begers procedure



freys procedure



Berns procedure

Frey introduced a procedure that combines duodenum-sparing resection of the pancreatic head, without formal division of the neck of the pancreas, combined with longitudinal pancreaticojejunostomy of the dorsal duct.[50] The Frey procedure appears to be an acceptable surgical alternative to achieve durable long-term pain relief and decrease opiate dependence in selected patients. In the experience of a number of series, 75% or greater success in relief of pain and gain of weight was seen after the Frey procedure.[51,52] For patients with an inflammatory head mass but small duct disease, Izbicki introduced a procedure that combines excavation of the pancreatic head with a V-shaped longitudinal wedge resection, followed by lateral decompressive pancreaticojejunostomy of the pancreatic body and tail.[53] Complete pain relief was reported in 92% of the patients, and improvements were seen in physical status, working ability, and emotional and social functioning to varying degrees.

TO SUMMARIZE

Chronic pancreatitis results in the progressive and irreversible destruction and replacement of the normal pancreatic parenchyma with fibrosis, ultimately leading to exocrine and, later, endocrine insufficiency. There is no simple unifying mechanism of pathogenesis. Rather, it is the interaction among risk factors including environmental exposure, genetic factors, and anatomic anomalies that appears to predispose to the development of chronic pancreatitis.

Recognition of chronic pancreatitis in its earliest stages continues to pose a challenge despite improvements in diagnostic testing and imaging. Treatment decisions should be guided by patient presentation but are hampered by the lack of consensus guidelines and by clinician bias. Therapeutic options include risk modification, analgesic therapy, diet, endoscopic therapy, and surgical therapy. Patients may be best treated in high-volume centers with radiologic, endoscopic, and surgical expertise, as well as an ancillary system of social workers, dieticians,and psychologists.

MATERIALS AND METHODS

All cases of Chronic calcific pancreatitis managed between 2011 August to February 2014 in our super specialty institute were analyzed

Age group, sex, occupation, presenting symptoms, co-morbid illness schedule type of surgeries performed & outcome were analyzed

INCLUSION CRITERIA

Patients with classical history and radiological charecteristics of chronic calcific pancreatitis

EXCLUSION CRITERIA

Patients with chronic calcific pancreatitis who are not willing to abstain from alcohol

Patients with poor performance status

INVESTIGATION DETAILS

Blood investigations including CBC, Liver function tests and RFT, CA 19-9, Viral markers.

USG abdomen – to look for pseudocyst

Portal Doppler : to look for associated portal hypertension

UGI Scopy: to look for extraneous impression and varices in cases of portal hypertension

CECT Abdomen & Pelvis

To look for calcification , head mass, stones in the duct and parenchyma and diameter of the head and associated complications in the form of psedocyst and peri splenic collaterls ,

CT- Angiography - To look for pseudo aneruysms around pancreas

MRCP and MRI- To look for status of CBD in cases presenting with jaundice and cholelithiasis.

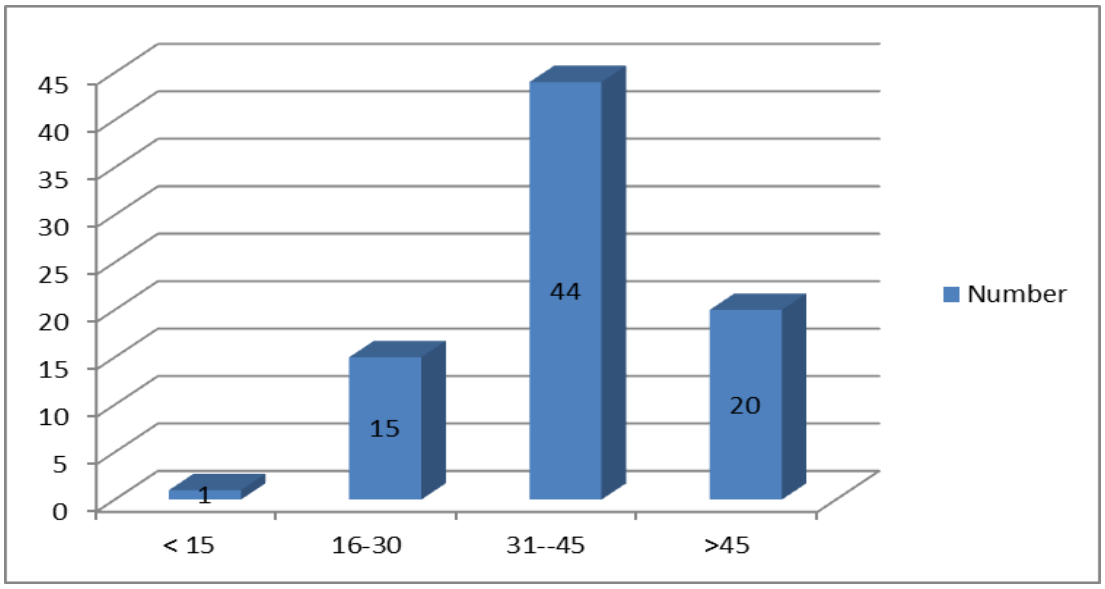
OBSERVATION AND RESULTS

A total number of 80 patients who were diagnosed to have chronic calcific pancreatitis , successfully manged were included in the study. The age of the patients varies between 13 to 58 yrs. Most of the patients were in their active earning period of life 30 to 45 yrs. (Tab 1)

Table-1: Age Groups

Age groups	Number
< 15	1
16-30	15
31—45	**44**
>45	20

Age distribution

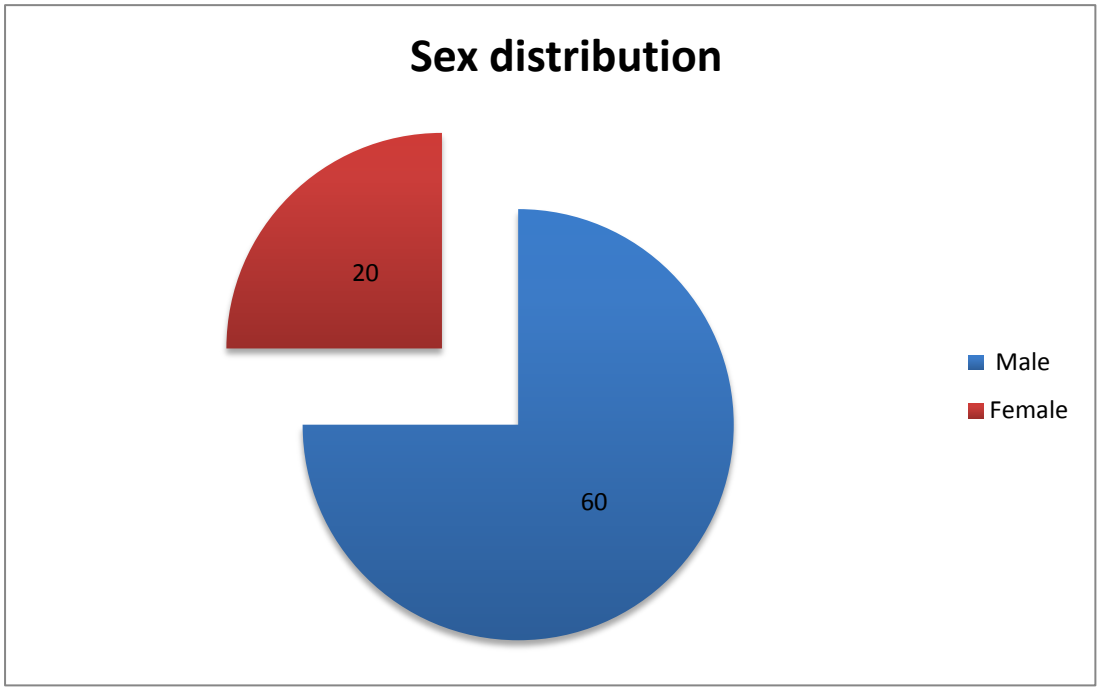


Out of 80 patients , 60 were male and 20 were female . (Tab2)

Tab 2:

Sex	Number	Percentage
Male	60	75%
Female	20	25%

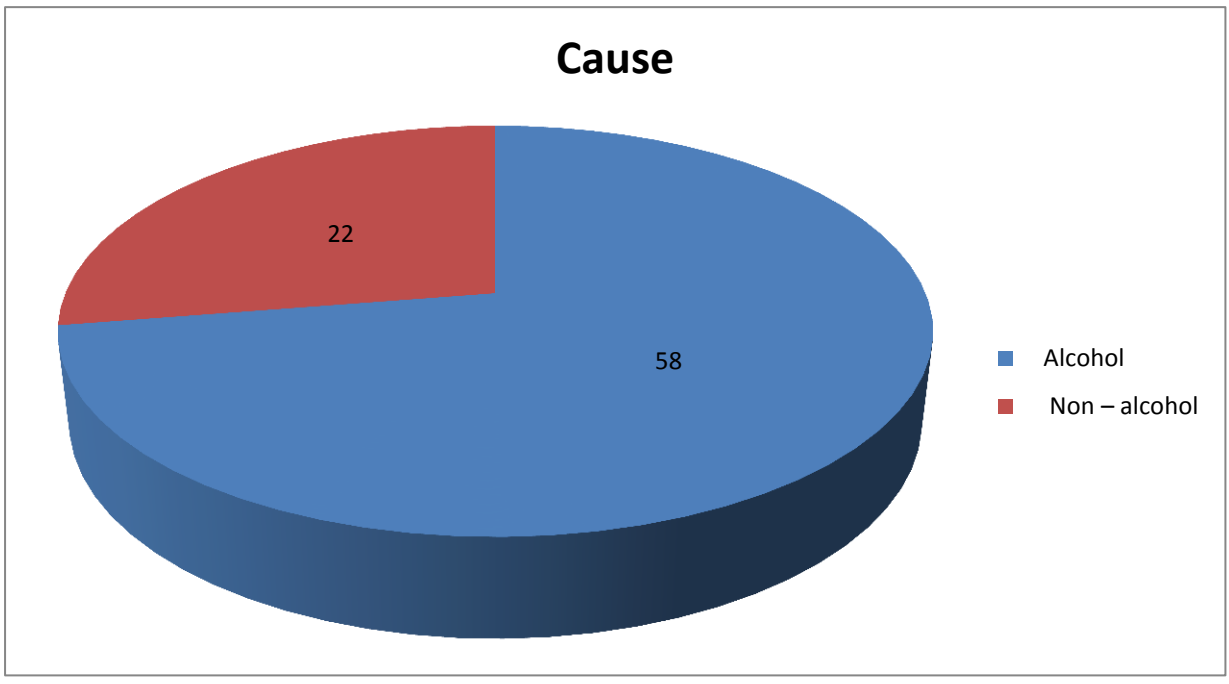
SEX DISTRIBUTION



Regarding the etiology of chronic calcific pancreatitis, alcohol was associated in 58 patients and 22 patients were considered to be tropical. (Tab3).

Tab 3:

Cause	Number	Percentage
Alcohol	58	72.5%
Non – alcohol	22	27.5%

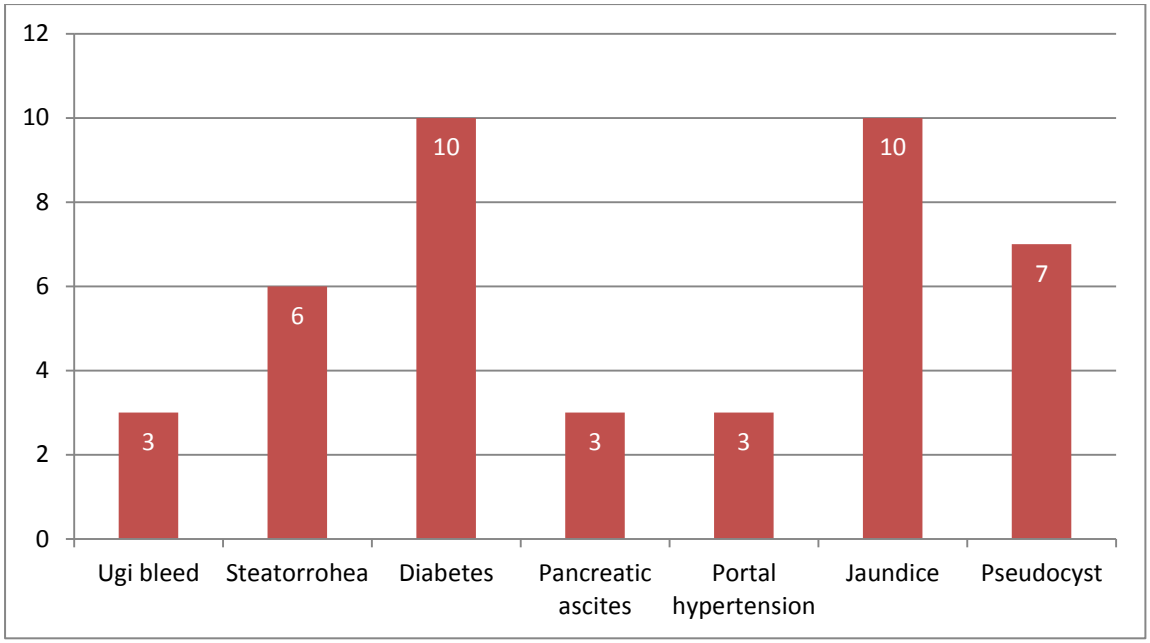


CLINICAL PRESENTATION:

Among the clinical presentations, all the patients were presented with abdominal pain, and the pain score was more than 8 for 68 patients and less than 8 for 12 patients . In addition to pain, the other clinical presentation were shown in (Tab 4)

Tab 4:

Ugi bleed	3
Steatorrohea	6
Diabetes	10
Pancreatic ascites	3
Portal hypertension	3
Jaundice	10
Pseudocyst	7



INVESTIGATIONS:

All patients underwent

Blood investigations including CBC, Liver function tests and RFT

USG abdomen – to look for psedocyst

Portal Doppler : to look for associated portal hypertension

UGI Scopy: to look for extraneous impression and varices in cases of portal hypertension

CECT Abdomen & Pelvis – to look for calcification , head mass, stones in the duct and parenchyma and diameter of the head and associated complications in the form of pseudocyst and peri splenic collaterals

MRI abdomen: to look for congenital anomalies like pancreas divisum.

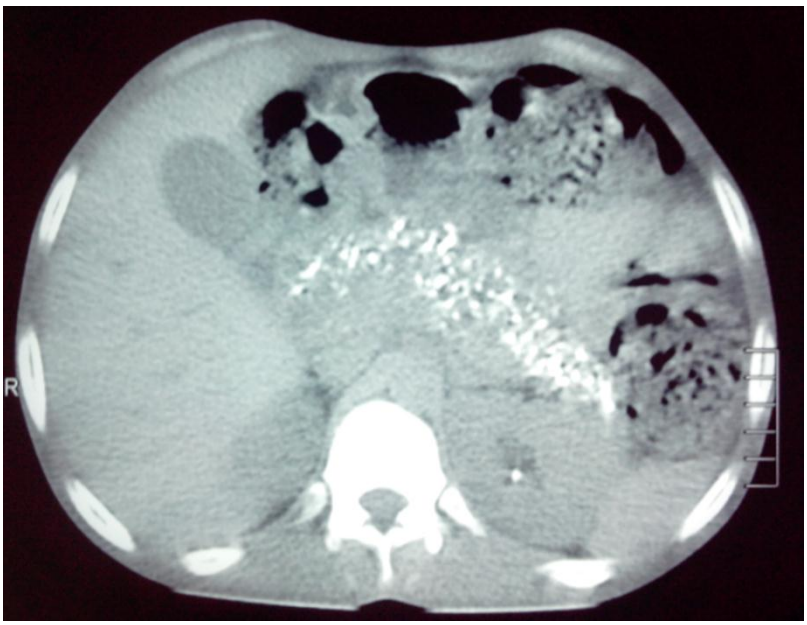


Fig A: Plain CT showing calcification

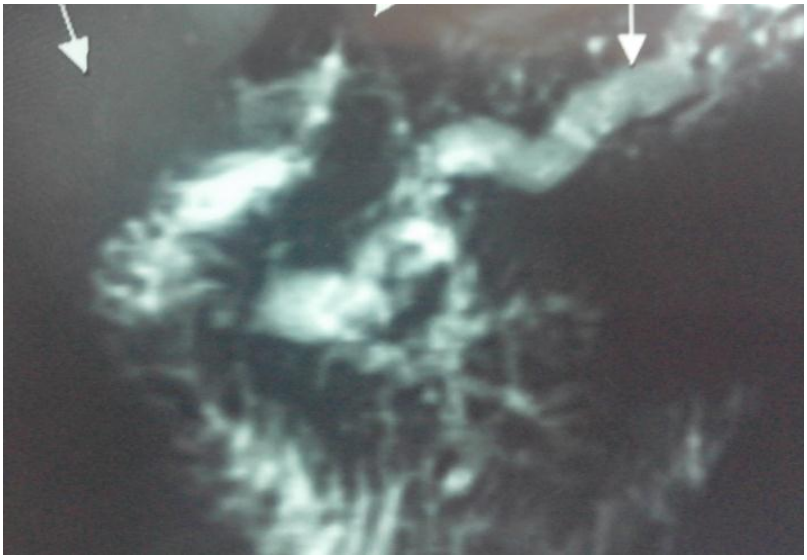


FIG:B

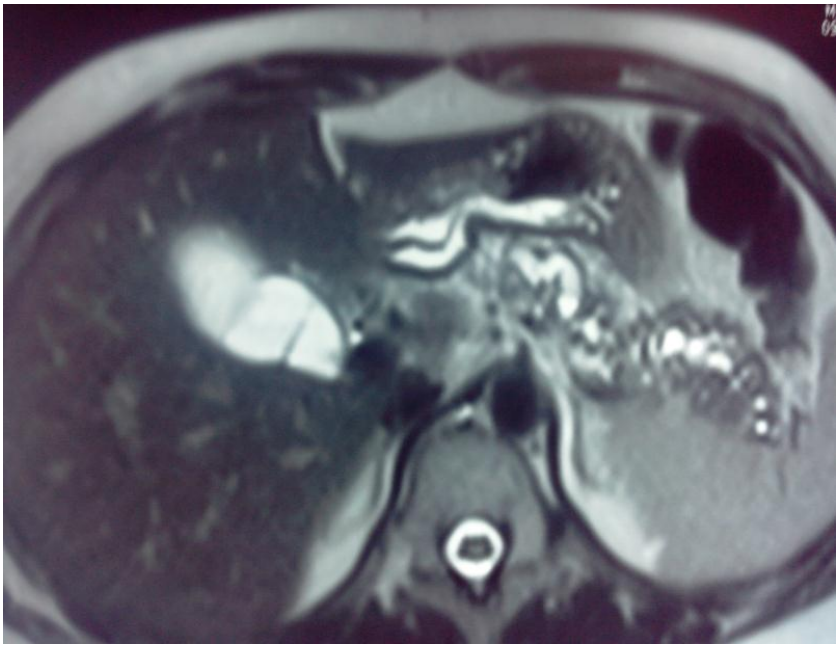


Fig :C

Fig B, C: MRI showing dilated main pamcreatic duct

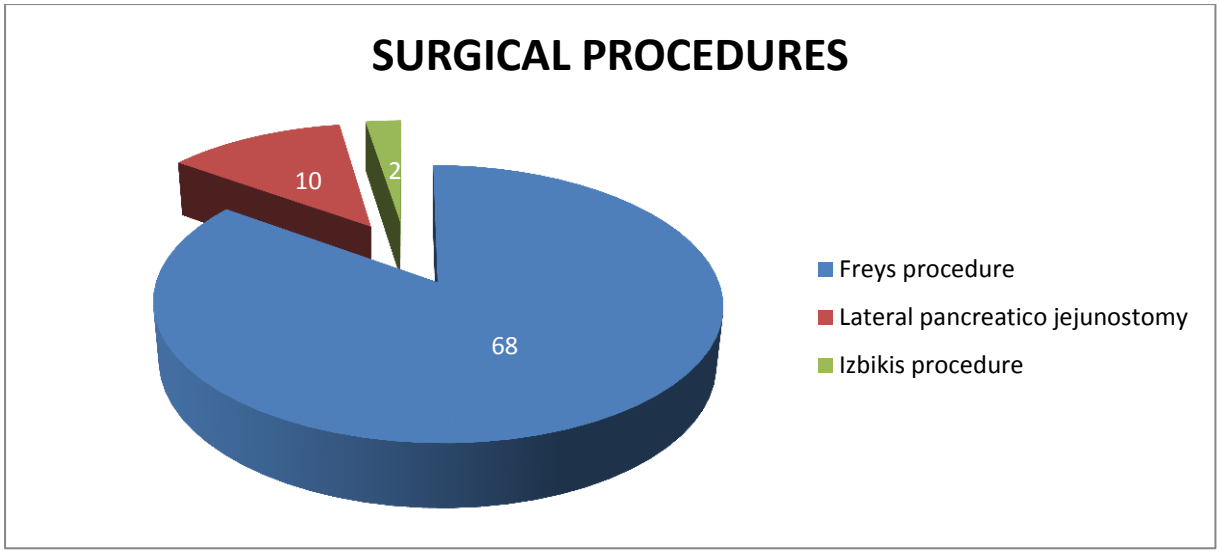
We found that , jaundice was evident in 10 patients, 3 patients were presented with portal hypertension, one patient was presented with pancreatico pleural fistula, one patient was presented with infected pseudocyst in the tail of pancreas

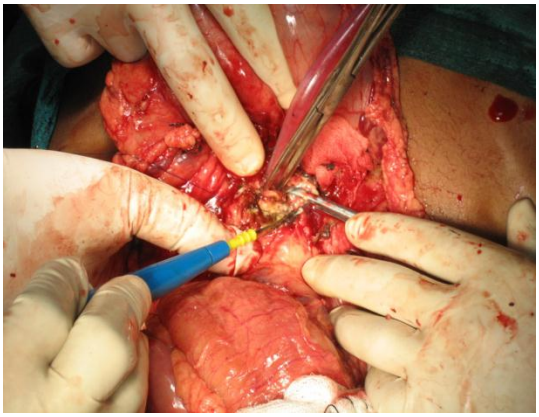
SURGICAL PROCEDURES

The patients have been chosen according to the diameter of the duct, presence of an inflammatory mass in the head region,and associated with other complications in the form pseudocyst, portal hypertension,jaundice.

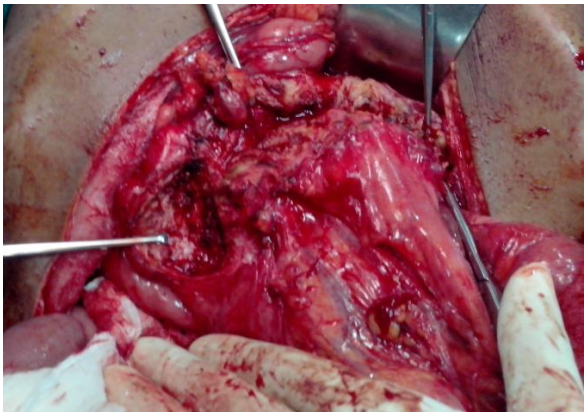
All the anastomosis was done using 2”0 prolene as a single layer anastomosis

Name of the procedure	Number
Freys procedure	68
Lateral pancreatico jejunostomy	10
Izbikis procedure	2

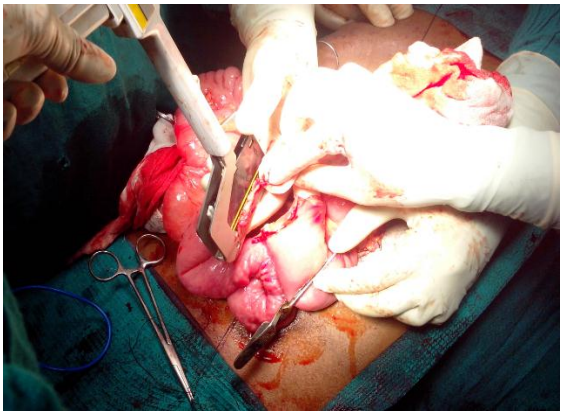




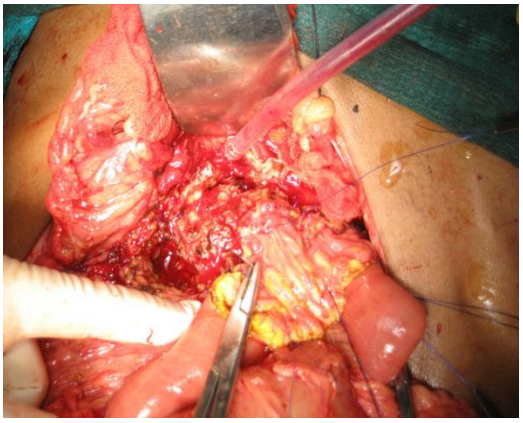
Removal of stones



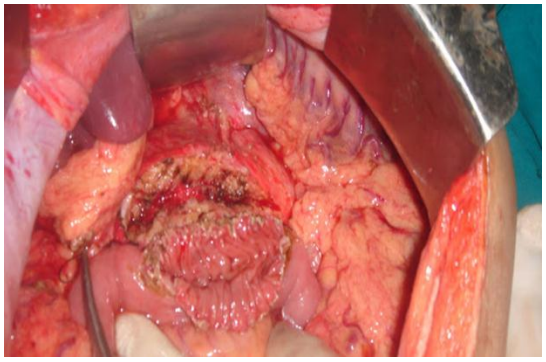
head coring& opening of duct



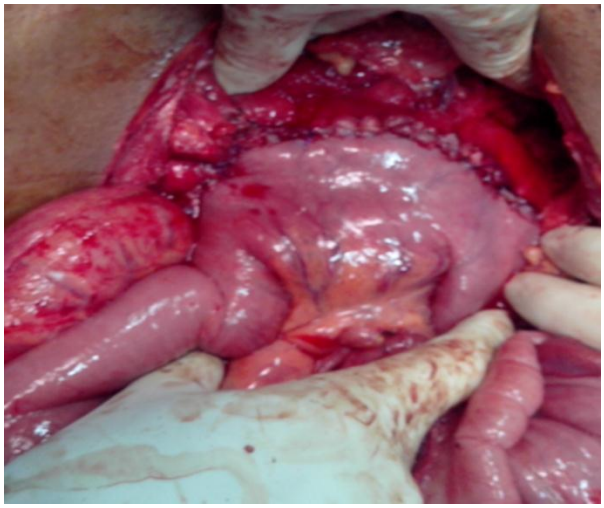
Creation of roux loop



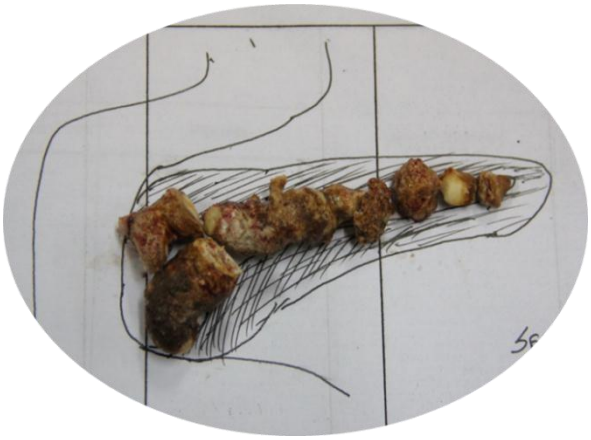
Anastomosing the jejunum



Anastomosing the jejunum with pancreas



Completed pancrteatico jejunostomy

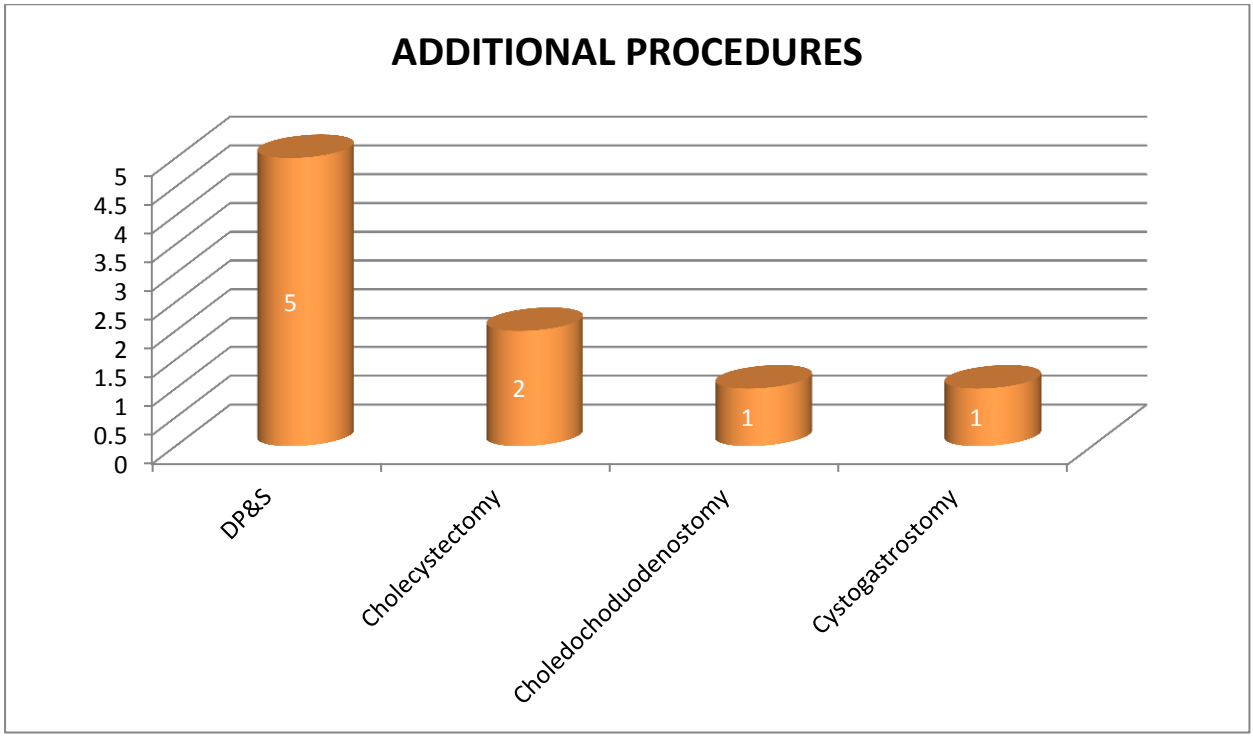


Removed stones

OTHER ASSOCIATED PROCEDURES

In addition to the standard procedures, we have performed other procedures to address the other complications.

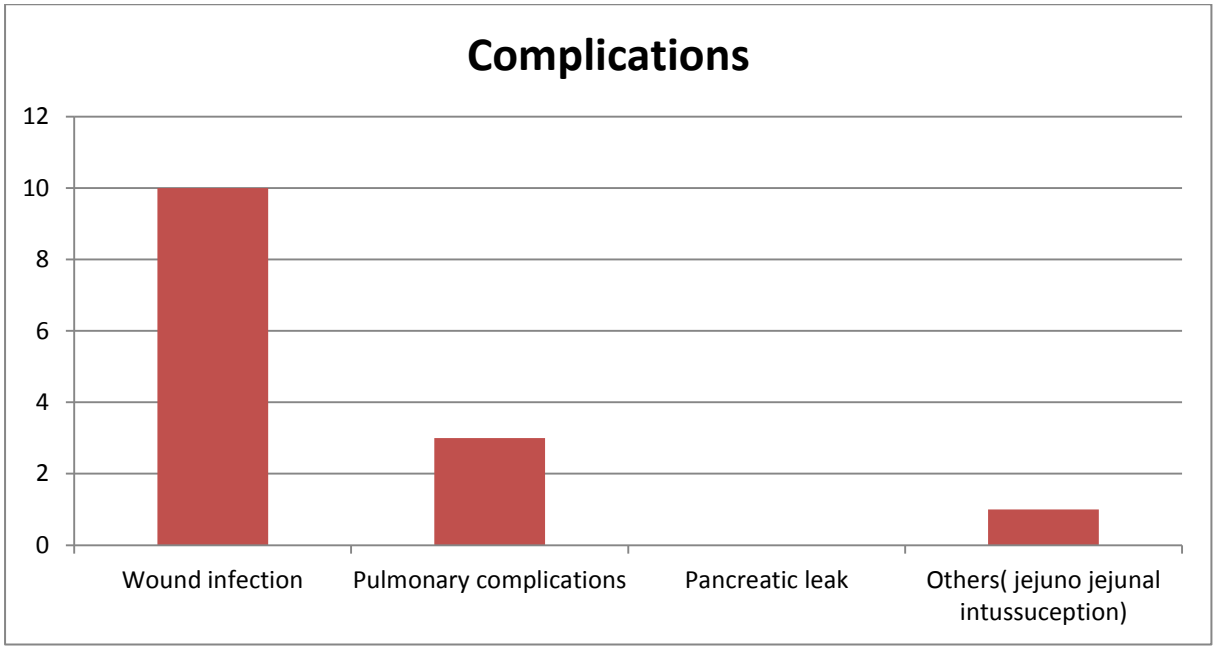
Name of the procedure	Number
Distal pancreatectomy& spleenectomy	5
Cholecystectomy	2
Choledochoduodenostomy	1
Cystogastrostomy	1



POST OP COMPLICATIONS

As like any other surgeries, the complication following the surgical procedures for chronic calcific pancreatitis are,

Complications	Number
Wound infection	10
Pulmonary complications	3
Pancreatic leak	0
Others(jejuno jejunal intussuception)	1



FOLLOW UP:

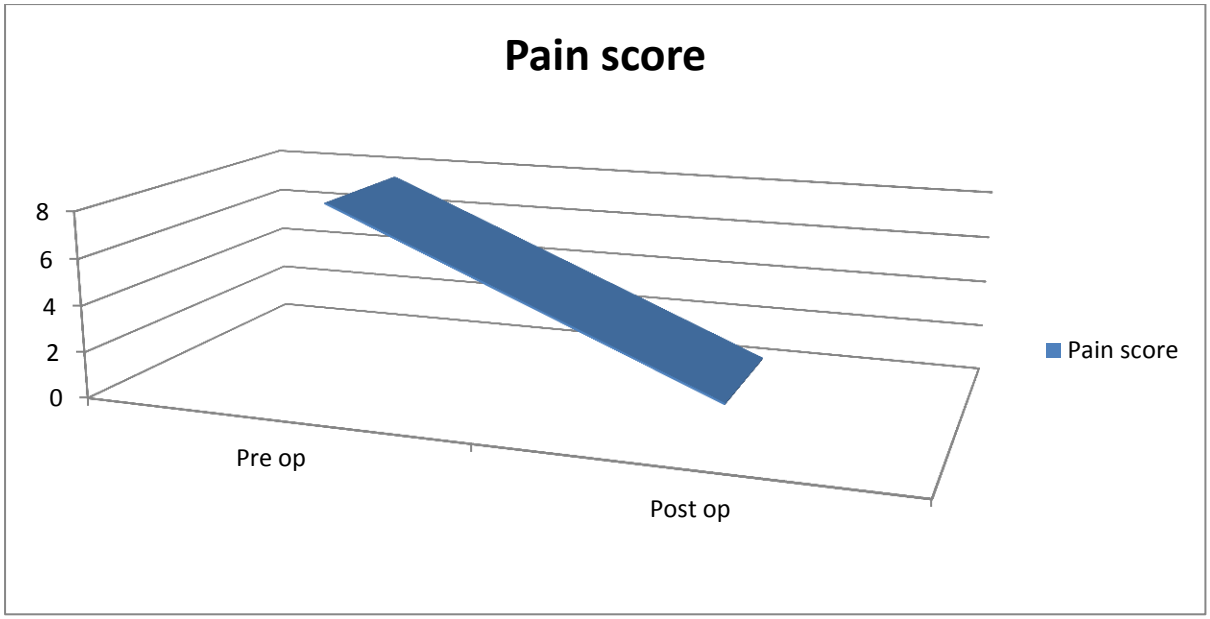
In the immediate follow up period, all the patients had pain relief drastically to the pain score of one and continues to be asymptomatic for a period of six months.

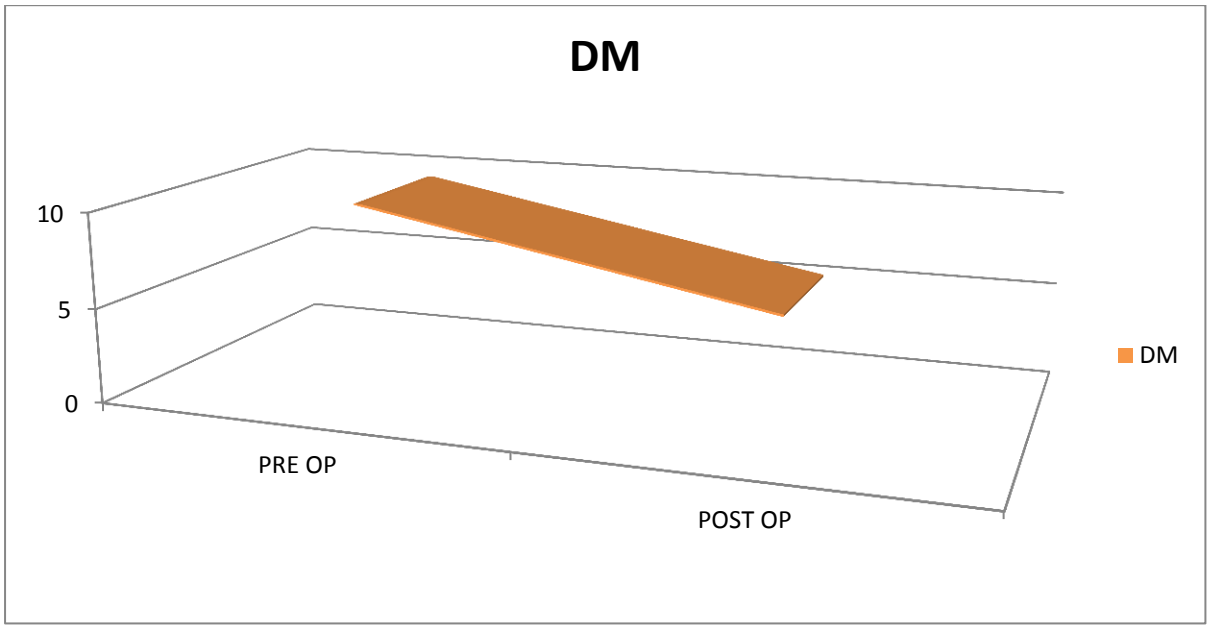
Three patients were re admitted with recurrence of pain due to the resumption of alcohol.

One patient who underwent lateral pancreatico jejunostomy was readmitted with gastric outlet obstruction and GJ was performed, the intra operative biopsy showed malignancy.

One patient was re admitted with jejuno jejunal intussception and he was managed successfully by resection and anastomosis.

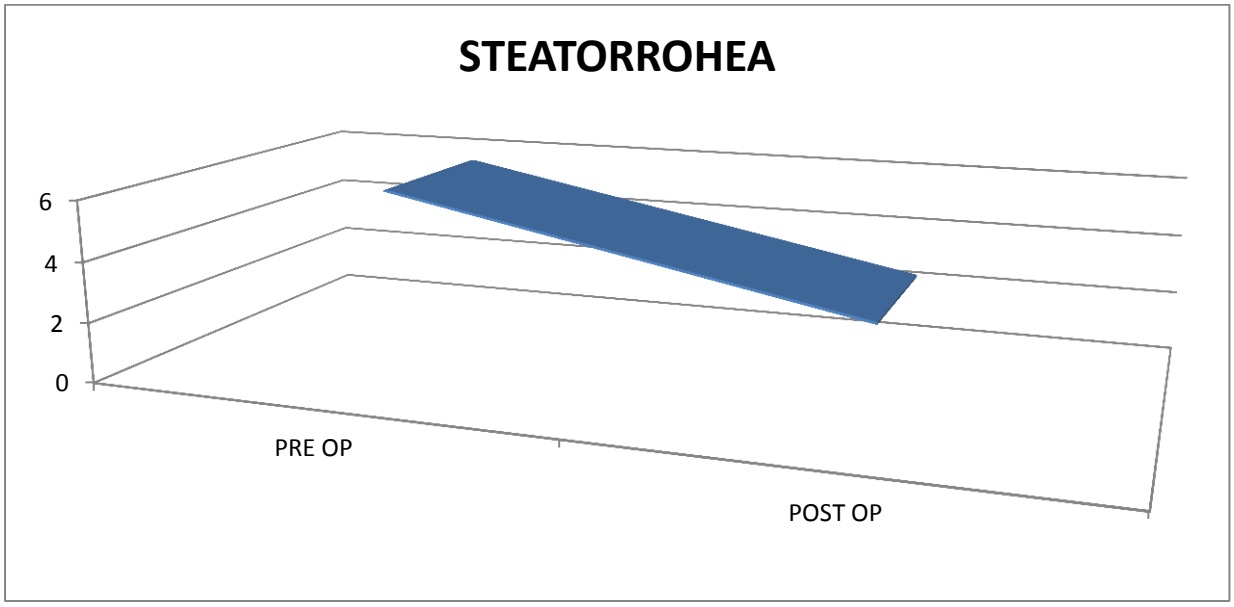
Follow up	Pre op	Post op
DM	10	6
Steatorrohea	6	3





The average pain score of the patient which was 8 pre operatively has come down to 1 in the immediate post op period.

Regarding the endocrine functions, the glycemic control was achieved in 6 out of 10 patients and tn terms of exocrine function, 3 out of 6 patients were under control.



DISCUSSION

Surgical intervention for chronic pancreatitis is commonly accepted as the most effective therapeutic option for pain control and management of complications.The main aim of the surgical treatment of CP is to alleviate severe pancreatic pain and to manage pancreatitis-related locoregional complications .Although lateral pancreatico jejunostomy(partingtons rochalle) which had been practised over 3 decades, it is clear that this is not a technique that solves all problems for patients with CP, especially those when there is no pancreatic duct dilatation or the disease is predominantly located in the head of the gland.

Many of the patients who underwent LPJ had recurrence of pain , which was often attributed to persistence or relapse of the disease in the pancreatic head. So the symptomatic relief which was the demand by most of the patients were not met. It is only the symptomatic relief that we are aiming to achieve.The principle of Frey's procedure is to decompress the branch ducts in the head of the pancreas, which is considered to be the pacemaker of the disease ("controller of inflammation,") [56] and to core out an inflammatory mass, which leads to locoregional complications. The mass in the pancreatic head region is excised almost in its entirety, leaving behind a bridge of pancreatic tissue about 1cm wide, while a rim of pancreas (5 to 10 mm) remains beside the duodenum and on the upper margin of the pancreatic head [53,54] The pancreatic neck above

the portal vein and superior mesenteric vein are left intact. The freys procedure is safer and easier to do by means of coring out of head mass as compared to PPPD. Prevention and recurence of complications is lower in PPPD as we remove the lower bile duct and duodenum. But freys procedure itself is sufficient for relieving jaundice in many of our cases. But regarding the post operative quality of life in terms of pain relief freys procedure gives much better life. Izbicki et al [55] performed a prospective randomized study of 61 patients comparing Frey's procedure and PPPD and Long-term Outcomes after Frey's Procedure concluded that Frey's procedure achieved equal pain relief and had a lower morbidity rate (19%) compared with PPPD (53%) and achieved better QOL scores (71 vs. 43) over a 2-year follow-up period.

Our observation and results supports the fact that freys procedure is considered to be the best procedure in patients with chronic calcific pancreatitis with a head mass. In terms complications in the form jaundice was also could be managed successfully with freys procedure itself. Three patients were presented with recurrence of pain due to resumption of alcohol in the post operative period were managed with celiac neurolysis and one patient was presented with gastric oultlet obstruction about 6months later was treated by gastrojejunostomy found to have malignancy during the post op period, which managed with celiac plexus neurolysis.. The incidence of these complications, requiring further treatment, after surgery is not necessarily high, even if alcohol

consumption is resumed. We found that , in our study out of 80 patients 26 patients were presented with pancreatitis related locoregional complications, 32.5% which is less as compared to the study by Neggi et al [15] .

Additional procedure had been performed to manage the locoregional complications like choledocho dudodenostomy, cystogastrostomy and distal pancreatectomy with spleenectomy, none of our patients developed complications in relation to the additional procedures ,several articles published that they may end up with complications [18,19].

CONCLUSIONS

In our study about 55% of the patients were within the age group of 30-45 which is the prime earning period of life thereby limiting the socio economic status of the family.

Males were predominant than females.(75% Vs25%)

Ethanol was the prime etiologic factor (72.5%)

Pain was the primary mode of presentation and about 32.5% were presented with locoregional complications of panceatitis.

Endocrine insufficiency was present in about 12.5% among that 7.5% of the patients had good glycemic control in immediate post op period.

Exocrine insufficiency was present in 7.5% of the patients, of which 3.5% had good symptomatic relief.

The results of the study in our institute, revealed the fact that the extended drainage procedure with coring out of tissue in the region of the head as proposed by Frey (resection and drainage) has high effectiveness in the treatment of pain , combined with little interference in the disease course (endocrine and exocrine function).

All the patients in our institute were operated by **a single layer anastomotic technique**, using 2" 0 prolene which is very cost effective, less time consuming and showing no difference in terms of post operative complications like pancreatic leak.

Frey's procedure should be considered as the primary operation in patients with disabling pain as a result of CP because it is safer, easier and presents less morbidity and **zero mortality** than alternative technique if it is performed in a high volume center.

The long-term results of pain relief are in excess of 90% and there may be functional benefit in terms of preservation of pancreatic function in the long term.

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CASE REPORT FORM

Name:
Age/sex:
Address:
Contact Numbers:
Occupation:
Body weight:

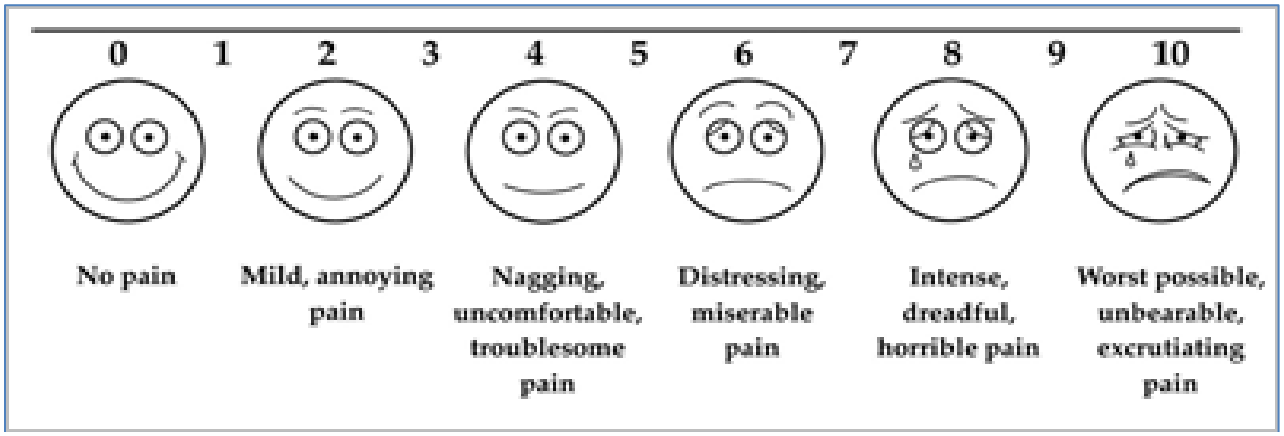
Personal habits:
Alcoholic:

- Duration
- Type of liquor
- Amount
- Manner

Smoker:

- Duration
- Type
- Number

Clinical Features:
Visual analog scale: Pain score: (Tick)



Duration of pain:
Character of pain:

Past H/o: Any surgery:

Symptoms	Tick		Signs	Tick
Steatorrhea			Thin built	
Diabetes			Splenomegaly	
Diarrhoea			Mass abdomen	
Hematemesis			Ascites	
Melena			Jaundice	
Weight loss				
Jaundice				

Diabetes:

- On Tablets
- On Insulin

Clinical examination

Investigations

TC- FBS: PPBS: Hemoglobin- Amylase- Lipase-
CA 19-9: Se albumin Bilirubin SGOT/SGPT PT /INR:
UGI SCOPY- PV Doppler- CECT abdomen- USG abdomen- MRCP-

Final diagnosis:

1. Tropical pancreatitis
2. Alcoholic pancreatitis
3. Anything else
4. Associate complications:
5. Diabetic

Management:

Surgery done:

Date of Surgery:

Procedure in nutshell:

Anastomosis technique:

Any other Important findings Associated:

Operative time:

Operative blood loss:

Intra Operative transfusion:

Intraop complications:

Post Op Events:

Blood transfusion:

Wound infection:

Ryles tube removal:
Oral started on:

Pain score after 1 week:

Blood sugar after 1 week:

Discharge Post op Day:

Biopsy no; Biopsy report:

Follow up:

After 1 month:

- Pain score
- Weight gain
- Diabetes- FBS/ PPBS
- Any other complaint

After 3 months:

- Pain score
- Weight gain

- Diabetes- FBS/ PPBS
- Any other complaint

After 6 months:

- Pain score
- Weight gain
- Diabetes- FBS/ PPBS
- Any other complaint

Information to Participants

Title: - **" ANALYSIS OF PRESENTATION, MANAGEMENT AND OUTCOME OF CHRONIC CALCIFIC PANCREATITIS IN A TERTIARY CARE CENTER "**

Principal Investigator: Dr.R.Rajkumar
Co-Investigator(if any):

Name of Participant:

Site :

You are invited to take part in this research/ study/procedures/tests. The information in this document is meant to help you decide whether or not to take part. Please feel free to ask if you have any queries or concerns.

What is the purpose of research?

Colorectal cancer is the third most frequent cancer in men and women. Surgery remains the mainstay of treatment which alone results in local recurrence in locally advanced tumors. Improved staging, introduction of multidisciplinary decision-making, refined surgery and appropriate use of preoperative radiotherapy, together with quality assessment by pathology and registries have all contributed to substantially lowering rates of local recurrence in rectal cancer from 30% to below 10%. Current standard for locally advanced rectal cancer includes preoperative chemoradiation followed by surgery. We want to test the efficacy and safety of a new _____ (drug / intervention / surgery /procedure/lab test) in this disease/condition.

We have obtained permission from the Institutional Ethics Committee.

The study design

Prospective and Retrospective study

Study Procedures

The study involves evaluation of Locally advanced Carcinoma Rectum treated with neoadjuvant therapy for which we will need tumor markers, barium enema colonoscopy, CECT Abdomen & Pelvis. The planned scheduled involve visits at _____,_____,____,

and_____(days/ weeks) after your initial visit. You will be required to visit the hospital _____ number of times during the study.

At each visit, the study physician will examine you. Some [blood / urine /imaging/clinical examination other] tests will be carried out at each visit. [… … ml of blood will be collected at each visit. Blood

collection involves prick with a needle and syringe.] These tests are essential to monitor your condition, and to assess the safety and efficacy of the treatment given to you.
In addition, if you notice any physical or mental change(s), you must contact the persons listed at the end of the document.

You may have to come to the hospital (study site) for examination and investigations apart from your scheduled visits, if required.

Possible risks to you – If any, Briefly mention

Possible benefits to you - If any, Briefly mention

Possible benefits to other people

The results of the research may provide benefits to the society in terms of advancement of medical knowledge and/or therapeutic benefit to future patients.

Confidentiality of the information obtained from you

You have the right to confidentiality regarding the privacy of your medical information (personal details, results of physical examinations, investigations, and your medical history). By signing this document, you will be allowing the research team investigators, other study personnel, sponsors, Institutional Ethics Committee and any person or agency required by law like the Drug Controller General of India to view your data, if required.
The information from this study, if published in scientific journals or presented at scientific meetings, will not reveal your identity.

How will your decision to not participate in the study affect you?

Your decision not to participate in this research study will not affect your medical care or your relationship with the investigator or the institution. You will be taken care of and you will not loose any benefits to which you are entitled.

Can you decide to stop participating in the study once you start?

The participation in this research is purely voluntary and you have the right to withdraw from this study at any time during the course of the study without giving any reasons. However, it is advisable that you talk to the research team prior to stopping the treatment/discontinuing of procedures etc.

Signature of Investigator
Date

Signature of Participant
Date

				clinical presentation													surgery done			intra op				post op				follow up	
S.NO	age/sex	diagnosis	abd. pain	back. Pain	bleed	jaundice	wt loss	ascites	pseudocyst	portal. HT	teatorhoea/DM	pain	palor	alcohol	calcification	ductal dia	procedure	others	d.o.surgery	blood loss	duration ⁀hrs⁀	⁀gm⁀	others	wound inf	blood trans	improval of drain	pain score	⁀1m⁀	at 6m
1	26/f	ccp	Y	N			Y					6				10	freys		17-01-14	100	2	4		y		3	1	1	1
2	24/f	ccp	y	y		y	y					8			y	8	freys		6/3/2014	100	3	6		y	y	5	1	1	1
3	36/m	ccp	y	y			y	y				8		y	y	7	lpj	dp+s		200	3	0		y	y		1	1	
4	40/m	ccp	Y	Y		Y	Y					7		Y	y	10	freys		11/1/2014	150	3	5				3	1	1	1
5	46/m	ccp	y	y					y						y	5	cons												
6	38/m	ccp	Y	Y			Y					8		Y	y	11	freys		6/1/2014	120	2	5				3	1	1	1
7	57/f	ccp	Y	Y			Y					9			y	10	freys		14-12-13	200	2.5	5				3	1	1	1
8	42/m	ccp	Y	Y			Y					9		Y	y	9	freys		5/12/2013	150	3	7				3	1	1	1
9	35/m	ac.on ch	y	Y	y		Y								y	5	cons												
10	15/m	ccp	Y	Y			Y			Y		8	Y	Y	y	9	freys		28-11-13	300	3	6			y	3	1	1	2
11	40/m	ccp	Y	Y			Y					9		Y	y	10	freys		28-11-13	300	2.5	6				3	1	1	1
12	48/m	ccp	Y	Y		Y	Y				st	8		Y	y	10	freys	dp+s	23-11-13	200		6		y		3	1	1	1
13	16/f	CCP	Y	Y			Y				dm	9			y	10	freys		7/11/2013	300	3	6				3	1	1	1
14	42/f	CCP	Y	N			Y					8			y	10	freys		31-10-13	100	3	7				3	1	1	1
15	22/f	CCP	Y	Y			Y					9			y	10	freys		29-10-13	200	3	7				3	1	1	1
16	36/M	CCP	Y	Y			Y		Y			9		Y	y	10	freys		29-10-13	100	3	6				3	1	1	1
17	42/m	ac.on ch	y	y				y							y	9	lpj	dp+s	28-09-13	300	3	0	pnem			3	1	1	1
18	34/m	CCP	Y	N			Y					9		Y	y	10	freys		30-09-13	200	3	6		y		3	1	1	2
19	34/m	CCP	Y	Y			Y				st	7		Y	y	10	freys		12/9/2013	300	3	6				4	1	1	1
20	53/m	CCP	Y	Y			Y			Y		9	Y	Y	y	11	freys		29-08-13	350	3	6			y	3	1	1	1
21	31/m	CCP	Y	Y			Y					7		Y	y	12	freys		29-08-13	150	3	7				3	1	1	1
22	30/m	CCP	Y	Y			Y			Y	dm	9	Y	Y	n	11	LPJ		22-08-13	200	3	7			y	3	1	1	1
23	45/m	CCP	Y	Y		y	y					9		Y	n	11	freys	chole	14-08-13	200	3	7				3	1	1	1
24	48/m	CCP	Y	Y			Y					9		Y	y	10	freys		8/8/2013	100	2.5	7				3	1	1	1
25	45/m	CP	Y	N		y	Y				dm	8			y	10	freys	chole	16-08-13	200	2.5	8				3	1	1	1
26	51/m	CCP	Y	Y			Y					8		Y	y	10	freys		5/8/2013	100	3	7		y		3	1	1	1
27	43/m	CCP	Y	Y			Y					9		Y	y	11	freys		2/8/2013	150	3	6				3	1	1	2
28	43/m	ac.on ch	y	Y			Y	y				8			y	8	lpj		4/8/2013	300		0				3	1	1	1
29	44/m	CCP	Y	Y			Y				st	8		Y	y	10	freys		10/7/2013	200	3	6				3	1	1	2
30	48/f	CCP	Y	Y			Y					8			y	6	freys		12/7/2013	300	3	6				3	1	1	1
31	41/m	CCP	Y	Y			Y					8		Y	y	10	LPJ		11/7/2013	200	3	6	pnem			3	1	1	1
32	44/f	CCP	Y	Y			Y		Y			8	Y		y	8	freys		23-05-13	200	3	6			y	3	1	1	1
33	44/m	CCP	Y	Y			Y				st	8	Y	Y	y	6	freys		9/5/2013	200	3	7				4	1	1	1
34	48/m	CCP	Y	Y	y	Y	Y					8		Y	y	9	freys	dp+s	4/5/2013	300	3	7				3	1	1	1
35	24/f	CCP	Y	Y			Y			Y		9	Y		y	9	LPJ		26-04-13	200	3	7				3	1	1	1
36	40/m	CCP	Y	Y			Y					8		Y	n	9	freys		21-04-13	200	3	7				3	1	1	1
37	58/m	CCP	Y	Y			Y				dm	9		Y	y	9	freys		15-04-13	200	3	6			y	3	1	1	1
38	44/m	CCP	Y	Y			Y					8	Y	Y	y	9	LPJ		7/3/2013	100	3	6		y		3	1	1	1
39	49/m	CCP	Y	N			Y		y			9		Y	y	9	freys		7/3/2013	200	3	6				3	1	1	1
40	35/f	CCP	Y	Y		Y	Y					9			y	9	freys		28-02-13	100	3	6				3	1	1	1
41	20/f	CCP	Y	N			Y					8			y	9	freys		28-02-13	200	3	5				3	1	1	1
42	43/m	CCP	Y	Y			Y				dm	9			y	9	freys		28-02-13	100	3	5				3	1	1	1
43	36	CCP	Y	Y			Y					8			y	11	freys		28-02-13	200	3	5				3	1	1	1
44	40/m	CCP	Y	Y			Y					9		Y	y	10	freys		14-02-13	100	3	5				3	1	1	1
45	26/m	CCP	Y	Y			Y					9			y	9	LPJ		7/2/2013	200	3	5				3	1	1	1
46	40/m	CCP	Y	Y	Y		Y			Y	dm	9	Y	Y	y	9	freys	dp+s	7/2/2013	100	3	4		y	y	3	1	1	1
47	22/f	CCP	Y	Y			Y					8			y	9	freys		3/1/2013	200	3	4				3	1	1	1
48	52/m	CCP	Y	Y			Y					9		Y	y	9	freys		27-12-12	100	3	7	pnem			4	1	1	1
49	48/m	CCP	Y	Y			Y		y		dm	7		Y	y	9	freys	CG	22-12-12	100	3	7				3	1	1	1
50	45/m	ccp	Y	N			Y					8		Y	y	9	freys		21-12-12	200	3	7				3	1	1	1
51	37/m	CCP	Y	Y		Y	Y					8		Y	y	9	freys		11/12/2012	100	3	7				3	1	1	1
52	26/f	CCP	Y	Y			Y					9			y	9	freys		29-11-12	200	3	5				3	1	1	1
53	33/m	CCP	Y	Y			Y					8		Y	y	3	IZBIKI		29-11-12	200	33	6				3	1	1	1
54	32/m	CCP	Y	Y			Y					8			n	9	freys		27-11-12	200	2.5	6				3	1	1	1
55	28/f	CCP	Y	Y			Y			Y		8	Y		y	9	freys		7/10/2012	150	3	7		y		3	1	1	1
56	30/m	CCP	Y	Y			Y					8		Y	y	10	freys		20-09-12	200	3	6				3	1	1	1
57	42/f	CCP	Y	Y		Y	Y				dm	8			y	11	freys		14-08-12	300	3	6				3	1	1	1
58	34/m	CCP	Y	N			Y					8		Y	y	11	freys		30-07-12	200	3	7				3	1	1	1
59	36/m	CCP	Y	Y			Y					8	Y	Y	n	10	freys		25-07-12	100	3	7		y		3	1	1	1

52	26/f	CCP	Y	Y			Y					9			y	9	freys		29-11-12	200	3	5				3	1	1	1
53	33/m	CCP	Y	Y			Y					8		Y	y	3	IZBIKI		29-11-12	200	33	6				3	1	1	1
54	32/m	CCP	Y	Y			Y					8			n	9	freys		27-11-12	200	2.5	6				3	1	1	1
55	28/f	CCP	Y	Y			Y			Y		8	Y		y	9	freys		7/10/2012	150	3	7		y		3	1	1	1
56	30/m	CCP	Y	Y			Y					8		Y	y	10	freys		20-09-12	200	3	6				3	1	1	1
57	42/f	CCP	Y	Y		Y	Y				dm	8			y	11	freys		14-08-12	300	3	6				3	1	1	1
58	34/m	CCP	Y	N			Y					8		Y	y	11	freys		30-07-12	200	3	7				3	1	1	1
59	36/m	CCP	Y	Y			Y					8	Y	Y	n	10	freys		25-07-12	100	3	7		y		3	1	1	1
60	33/m	ccp	Y	Y			Y					8		Y	y	9	freys		23-07-12	200	3	6				3	1	1	2
61	58/m	ccp	Y	Y			Y				st	8		Y	y	8	freys		17-07-12	100	3	7			y	3	1	1	1
62	37/m	ccp	Y	Y			Y				dm	8		Y	y	9	freys		16-07-12	200	3	6	jj int			3	1	1	1
63	15/f	ccp	Y	Y			Y		Y			8			y	9	freys		3/7/2012	100	2.5	7				3	1	1	1
64	30/m	ccp	Y	Y		Y	Y					9			y	9	freys		14-05-12	200	3	6				3	1	1	1
65	15/f	ccp	Y	Y			Y			Y		9	Y		y	9	freys		3/5/2012	300	3	7				3	1	1	1
66	47/m	ccp	Y	Y			Y					9		Y	y	9	LPJ		12/7/2012	200	3	6				3	1	1	1
67	25/m	ccp	Y	Y			Y				st	9			y	9	freys		8/3/2012	100	3	6		y		4	1	1	1
68	48/m	ccp	Y	Y			Y					9		Y	n	10	freys		6/3/2012	200	3	7				3	1	1	1
69	42/m	ccp	Y	Y			Y					9	+	Y	y	9	freys		6/3/2012	200	3	8				3	1	1	1
70	36/f	ccp	Y	Y			Y					9			y	9	freys		23-02-12	300	3	8				3	1	1	1
71	40/m	ccp	Y	Y			Y					9		Y	y	9	freys		17-02-12	150	3	6				3	1	1	1
72	13/m	ccp	Y	Y			Y					9			y	9	freys		24-01-12	200	3	6				3	1	1	1
73	30/m	ccp	Y	Y			Y					9		Y	y	9	freys		15-12-11	100	3	6				3	1	1	1
74	45/m	ccp	Y	Y			Y			Y	dm	8	Y	Y	y	3	IZBIKI		19-11-11	150	3	6				3	1	1	1
75	32/m	ccp	Y	Y			Y		Y			7	Y	Y	n	10	freys		10/11/2011	200	3	6		y		3	1	1	1
76	30/f	ccp	Y	Y			Y					8			y	10	freys		8/11/2011	100	2.5	7				3	1	1	1
77	46/m	ccp	Y	Y		y	Y			Y		8		Y	y	9	freys	CDD	22/09/11	200	3	7				3	1	1	1
78	34/m	ccp	Y	Y			Y					9		Y	y	9	freys		6/9/2011	100	3	6				3	1	1	1
79	35/F	ccp	Y	Y			Y					9	Y		y	9	freys			200	3	6		y		3	1	1	1
80	23/f	ccp	y	y			y					8			y		lpj			200	3	7				2	1	1	1